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Prostacyclin-induced vasodilation in rabbit heart is mediated by ATP-sensitive potassium channels.

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We tested the hypothesis that prostacyclin and its stable analogue iloprost act as agonists of ATP-sensitive potassium channels (KATP) to induce vasodilation of the coronary circulation. The selective blocker of KATP, glibenclamide, was used as a probe for vasodilation mediated by KATP in saline-perfused rabbit hearts (constant flow, Langendorff preparation). Glibenclamide (10-300 nM) significantly increased coronary perfusion pressure and inhibited vasodilation induced by iloprost (1-30 nM), prostacyclin (10 nM), adenosine (0.3 microM), and cromakalim (0.1 microM), a known agonist of KATP. This potassium channel antagonist also inhibited vasodilation of rabbit hearts in response to 10 nM bradykinin in the presence of an inhibitor of nitric oxide synthase (30 microM NG-nitro-L-arginine). Because bradykinin-induced vasodilation is mediated by prostacyclin released from endothelial cells when nitric oxide synthesis is inhibited, these data indicate that glibenclamide is also effective against endogenous prostacyclin. The inhibitory effects of glibenclamide were selective: vasodilation induced by sodium nitroprusside (1-10 microM) or acetylcholine (1 microM) were not inhibited by this potassium channel antagonist. In addition, basal and bradykinin-stimulated release of 6-ketoprostaglandin F1 alpha was not affected by this antagonist of KATP. Glibenclamide also did not inhibit the activation of adenylate cyclase, as indicated by its lack of effect on adenosine 3',5'-cyclic monophosphate accumulation induced by iloprost (10 nM-1 microM) in bovine coronary arterial segments, a tissue in which iloprost-induced vascular smooth muscle relaxation is inhibited by glibenclamide.

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